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NEWS 10 JUN 02
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                INPADOC
NEWS 11
        JUN 26
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                and display fields
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        JUN 28
                Price changes in full-text patent databases EPFULL and PCTFULL
NEWS 13 JUl 11 CHEMSAFE reloaded and enhanced
NEWS 14 JUl 14 FSTA enhanced with Japanese patents
NEWS 15
        JUl 19 Coverage of Research Disclosure reinstated in DWPI
NEWS 16 AUG 09
                INSPEC enhanced with 1898-1968 archive
        AUG 28 ADISCTI Reloaded and Enhanced
NEWS 17
NEWS 18 AUG 30 CA(SM)/CAplus(SM) Austrian patent law changes
NEWS EXPRESS
             JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
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=> s somatostatin or neurotensin or penetratine or bombensin

19356 SOMATOSTATIN

146 SOMATOSTATINS

19365 SOMATOSTATIN

(SOMATOSTATIN OR SOMATOSTATINS)

4752 NEUROTENSIN

27 NEUROTENSINS

4755 NEUROTENSIN

(NEUROTENSIN OR NEUROTENSINS)

0 PENETRATINE

1 PENETRATINES

1 PENETRATINE

(PENETRATINE OR PENETRATINES)

1 BOMBENSIN

L1 23282 SOMATOSTATIN OR NEUROTENSIN OR PENETRATINE OR BOMBENSIN

=> s acridine or porphyrin or ellipticine or phenantroline or carbazole or benzimidazole or daunorubicine or epirubicine or mixoxantrone

17981 ACRIDINE

1711 ACRIDINES

18378 ACRIDINE

(ACRIDINE OR ACRIDINES)

35685 PORPHYRIN

24812 PORPHYRINS

41961 PORPHYRIN

(PORPHYRIN OR PORPHYRINS)

1033 ELLIPTICINE

147 ELLIPTICINES

1057 ELLIPTICINE

(ELLIPTICINE OR ELLIPTICINES)

171 PHENANTROLINE

5 PHENANTROLINES

174 PHENANTROLINE

(PHENANTROLINE OR PHENANTROLINES)

16646 CARBAZOLE

2183 CARBAZOLES

17214 CARBAZOLE

(CARBAZOLE OR CARBAZOLES)

23371 BENZIMIDAZOLE

```
5898 BENZIMIDAZOLES
         24718 BENZIMIDAZOLE
                 (BENZIMIDAZOLE OR BENZIMIDAZOLES)
            42 DAUNORUBICINE
            16 EPIRUBICINE
             0 MIXOXANTRONE
L2
        102010 ACRIDINE OR PORPHYRIN OR ELLIPTICINE OR PHENANTROLINE OR CARBAZO
               LE OR BENZIMIDAZOLE OR DAUNORUBICINE OR EPIRUBICINE OR MIXOXANTR
              ONE
=> s 12 and 12
       102010 L2 AND L2
=> s 12 and 11
            53 L2 AND L1
T.4
=> s conjugat? or coupl? or link? or combin?
        225632 CONJUGAT?
        783227 COUPL?
        466608 LINK?
       1115681 COMBIN?
1.5
       2438342 CONJUGAT? OR COUPL? OR LINK? OR COMBIN?
=> s 15 and 14
           29 L5 AND L4
L6
=> s 16 not py>1999
       7078308 PY>1999
             1 L6 NOT PY>1999
1.7
=> d ibib
    ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
L7
ACCESSION NUMBER:
                        1989:625888 CAPLUS
DOCUMENT NUMBER:
                         111:225888
                         Enprostil reduces the increase of gastric corpus
TITLE:
                         mucosal mass induced by the hydrogen-potassium-
                         stimulated adenosine triphosphatase inhibitor BY
                         831-78 in the rat
AUTHOR(S):
                         Inauen, W.; Rohner, C.; Koelz, H. R.; Herdmann, J.;
                         Schuerer-Maly, C. C.; Varga, L.; Halter, F.
CORPORATE SOURCE:
                         Gastrointest. Unit, Univ. Hosp., Bern, 3010, Switz.
SOURCE:
                         Gastroenterology (1989), 97(4), 846-52
                         CODEN: GASTAB; ISSN: 0016-5085
                         Journal
DOCUMENT TYPE:
LANGUAGE:
                         English
=> d abs kwic
     ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN
     It was determined if enprostil, a synthetic PGE2 derivative, might inhibit
AB
     release and the trophic effects on gastric oxyntic mucosa induced by
     prolonged treatment with an inhibitor of H+-K+-stimulated ATPase, the
     substituted benzimidazole BY 831-78. Rats were treated
     intragastrically with enprostil (1 or 15 \mu g/kg b.i.d.), BY 831-78 (15
     µmol/kg once daily), the combination of enprostil and BY
     831-78, ranitidine (300 µmol/kg b.i.d.), and placebo. Plasma gastrin
     and somatostatin levels and gastric acid secretion were measured
     during a 1-day treatment in animals fitted with chronic gastric fistulas
     and repeatedly during 9 wk of treatment in intact rats. Despite
     inhibiting acid secretion, enprostil did not increase plasma gastrin.
```

When combined with BY 831-78, enprostil transiently reduced the

BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137 pmol/L.12h) in fasted rats with fistulas, but failed to prevent the marked hypergastrinemia following 9 wk of treatment with BY 831-78 (717 vs. 731 pmol/L) in intact rats. However, enprostil reduced the BY 831-78-induced increase of oxyntic mucosal volume (458 vs. 567 mm3), whereas BY 831-78 prevented the enprostil-induced increase of antral mucosal volume (42 vs. 56 mm3). Apparently, some of the trophic effects induced by a H+,K+-ATPase inhibitor are not exclusively governed by gastrin. . . . and the trophic effects on gastric oxyntic mucosa induced by prolonged treatment with an inhibitor of H+-K+-stimulated ATPase, the substituted benzimidazole BY 831-78. Rats were treated intragastrically with enprostil (1 or 15 µg/kg b.i.d.), BY 831-78 (15 µmol/kg once daily), the combination of enprostil and BY 831-78, ranitidine (300 µmol/kg b.i.d.), and placebo. Plasma gastrin and somatostatin levels and gastric acid secretion were measured during a 1-day treatment in animals fitted with chronic gastric fistulas and repeatedly during 9 wk of treatment in intact rats. Despite inhibiting acid secretion, enprostil did not increase plasma gastrin. When combined with BY 831-78, enprostil transiently reduced the BY 831-78-induced increase of integrated plasma gastrin (1375 vs. 2137 pmol/L.12h) in fasted. 51110-01-1, Somatostatin RL: BIOL (Biological study) (secretion of, ATPase inhibitor and PGE2 analog effect on, gastrin in relation to) => s 16 not py>2000 6188416 PY>2000 2 L6 NOT PY>2000 => s 18 not 17 1 L8 NOT L7 => d ibib abs kwic ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN ACCESSION NUMBER: 2000:690483 CAPLUS DOCUMENT NUMBER: 133:361093 Ligand-induced internalization of neurotensin TITLE: in transfected COS-7 cells: differential intracellular trafficking of ligand and receptor AUTHOR(S): Vandenbulcke, Franck; Nouel, Dominique; Vincent, Jean-Pierre; Mazella, Jean; Beaudet, Alain Montreal Neurological Institute, McGill University, CORPORATE SOURCE: Montreal, QC, H2A 2B4, Can. SOURCE: Journal of Cell Science (2000), 113(17), 2963-2975 CODEN: JNCSAI; ISSN: 0021-9533 PUBLISHER: Company of Biologists Ltd. DOCUMENT TYPE: Journal LANGUAGE: English The neuropeptide neurotensin (NT) is known to be internalized in a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first

found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear

AΒ

IT

1.9

1.9

AB

compartment identified as the Trans-Golgi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.

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REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Ligand-induced internalization of neurotensin in transfected COS-7 cells: differential intracellular trafficking of ligand and receptor

- The neuropeptide neurotensin (NT) is known to be internalized in AΒ a receptor-mediated fashion into its target cells. To gain insight into the mechanisms underlying this process, we monitored in parallel the migration of the NT1 neurotensin receptor subtype and a fluorescent analog of NT (fluo-NT) in COS-7 cells transfected with a tagged NT1 construct. Fluo-NT internalization was prevented by hypertonic sucrose, potassium depletion and cytosol acidification, demonstrating that it proceeded via clathrin-coated pits. Within 0-30 min, fluo-NT accumulated together with its receptor in Acridine Orange-pos., acidic organelles. These organelles concentrated transferrin and immunostained pos. for rab 5A, therefore they were early endosomes. After 30-45 min, the ligand and its receptor no longer colocalized. Fluo-NT was first found in rab 7-pos. late endosomes and later in a nonacidic juxtanuclear compartment identified as the Trans-Golqi Network (TGN) by virtue of its staining for syntaxin 6. This juxtanuclear compartment also stained pos. for rab 7 and for the TGN/pericentriolar recycling endosome marker rab 11, suggesting that the ligand could have been recruited to the TGN from either late or recycling endosomes. By that time, internalized receptors were detected in Lamp-1-immunoreactive lysosomes. These results demonstrate that neurotensin/NT1 receptor complexes follow a recycling cycle that is unique among the G protein-coupled receptors studied to date, and provide the first evidence for the targeting of a nonendogenous protein from endosomes to the TGN.
- ST neurotensin complex NT1 receptor endocytosis intracellular trafficking
- IT Organelle

(coated pit; neurotensin internalization via NT1 receptors
proceeds via clathrin-coated pits)

IT Endosome

(internalized neurotensin/NT1 receptor complexes are initially targeted to endosomes upon import)

IT Biological transport

(intracellular; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Lysosome

(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Neurotensin receptors

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT Endocytosis

(receptor-mediated; neurotensin internalization via NT1
receptors proceeds via clathrin-coated pits)

IT Organelle

(trans-Golgi network; neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

IT 39379-15-2, Neurotensin

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(neurotensin internalized via NT1 receptors is recruited to trans-golgi network whereas receptors are targeted to lysosomes for degradation)

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